Common celiacomesenteric trunk: Aneurysmal and occlusive disease

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Eighteen patients (14 men, 4 women), ages 24 to 77 years, with a common celiacomesenteric trunk (CMT) were treated between 1965 and 2004 at the University of Michigan. Four patients had CMT aneurysmal or occlusive disease that led to operative treatment. Pertinent arteriographic findings in these 4 patients included a CMT aneurysm (n = 2), an occluded proximal CMT (n = 1), and a type III aortic dissection that was compressing the CMT (n = 1). Therapy in these 4 patients included placement of a polytetrafluoroethylene bypass graft from the supraceliac aorta to the CMT (n = 2) or a Dacron bypass graft from a thoracoabdominal bypass to the CMT (n = 1), and endovascular fenestration of the septum between the true and false lumens of an aortic dissection at the level of the CMT (n = 1). (J Vasc Surg 2004;40:1040-3.)

Anomalies of the celiac trunk and mesenteric arteries have been well-established.[1,2] The vascular variations most often affect the origins of the right or left hepatic arteries.[2-4] A celiacomesenteric trunk (CMT), with the celiac and superior mesenteric arteries having a common origin from the aorta, accounts for less than 1% of all splanchnic artery anomalies, and is estimated to have an incidence of 0.25%.[1,3-5] To date only 6 patients with CMT disease have been reported in 5 separate series.[6-11] The present report details a unique experience with 4 patients with aneurysmal and occlusive disease of a CMT who were treated at the University of Michigan. The therapeutic options and long-term outcomes after interventions in these patients provide insight into the complexity of intrinsic CMT disease.

METHODS

A retrospective review was performed of all patients undergoing visceral arteriography from 1965 to 2004. Arteriograms for patients with a reported diagnosis of CMT were evaluated. In addition, all records for patients treated at the University of Michigan from 1965 to 2004 with a discharge diagnosis including CMT were reviewed. The study was approved by the University of Michigan Institutional Review Board for human subjects (2001-0797).

Eighteen patients (14 men, 4 women) with CMT were treated. Median patient age was 59 years (range, 24 to 77 years). CMT was diagnosed at arteriography in all but 1 patient, in whom CMT was recognized intraoperatively. Four patients had aneurysmal or occlusive disease of the CMT, and form the basis of this report

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CASE REPORTS

Case 1. The first patient had weight loss and postprandial abdominal pain consistent with intestinal angina. Arteriography documented an occluded CMT (Fig 1, A). Successful revascularization was performed with placement of a polytetrafluoroethylene (PTFE) bypass graft originating from the distal thoracic aorta (Fig 1, B and C). The patient regained 70 lbs, and has remained without abdominal pain 4 years postoperatively.

Case 2. The second patient had hypertension refractory to medical therapy and was found to have a supra-CMT aortic aneurysm and a CMT aneurysm. Both right and left renal arteries arose from the CMT aneurysm. The patient underwent resection of the aortic aneurysm with a thoracoabdominal aortoaortic interposition graft, an aortoceliac artery Dacron bypass from the former graft, and revascularization of both renal arteries. The hypertension resolved, and the patient remained symptom-free for 16 years before dying of a cardiac event and stroke.

Case 3. The third patient had abdominal pain and hypertension, and was also found to have a tortuous upper abdominal aorta and a CMT aneurysm (Fig 2, A). The CMT aneurysm also gave rise to the right renal artery. The patient received an aortoaortic interposition PTFE graft to replace the tortuous upper abdominal aorta, a thoracic aorto-CMT PTFE bypass graft (Fig 2, B and C), and an aortorenal bypass graft with saphenous vein. The patient remains symptom free at 4 years.

Case 4. The fourth patient, with acute type III aortic dissection, had abdominal pain and arteriographic evidence of compromise of intestinal blood flow involving the CMT (Fig 3, A). Endovascular fenestration of the septum between the 2 lumens and stenting of the aortic true lumen improved flow to the CMT and its branches (Fig 3, B). The patient has remained without symptoms at 3 years.

Fourteen additional patients were found to have an incidental CMT. Thirteen of these anomalies were discovered at visceral arteriography performed for a number of indications, including presentation with nonvascular gastrointestinal symptoms (n = 6), anatomic delineation for hepatic artery chemotherapy infusion

Fig 1. A, Lateral aortogram demonstrates an occluded celiacomensenteric trunk (CMT; *arrow*). **B,** Aortogram after aorto-CMT bypass with polytetrafluoroethylene graft *(arrow)* demonstrates antegrade filling of CMT. **C,** Selective arteriogram of graft reveals hepatic, splenic, and superior mesenteric artery branches *(arrows)* arising from CMT.

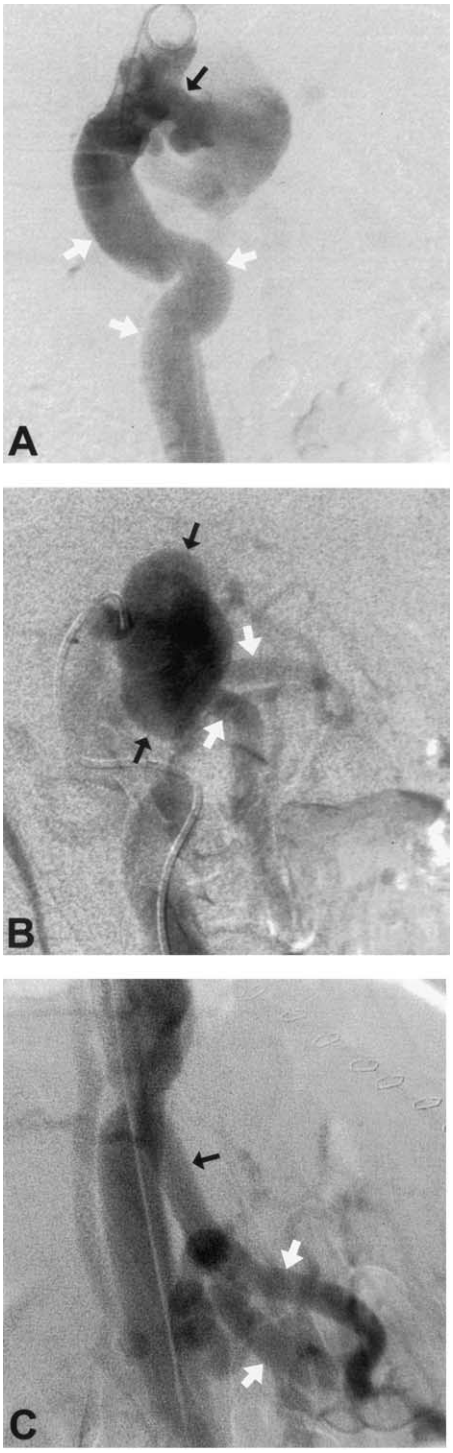

Fig 2. A, Initial lateral arteriogram reveals a celiacomensenteric trunk (CMT; *black arrow*) originating from a tortuous abdominal aorta *(white arrows)*. **B,** Selective CMT injection reveals a large CMT aneurysm *(black arrows)* and exiting celiac and superior mesenteric arteries *(white arrows)*. **C,** Postoperative aortogram demonstrates replacement of the tortuous aorta with a polytetrafluoroethylene interposition graft and a patent polytetrafluoroethylene aorto-CMT bypass *(black arrows)* with celiac and superior mesenteric branches *(white arrows)*.

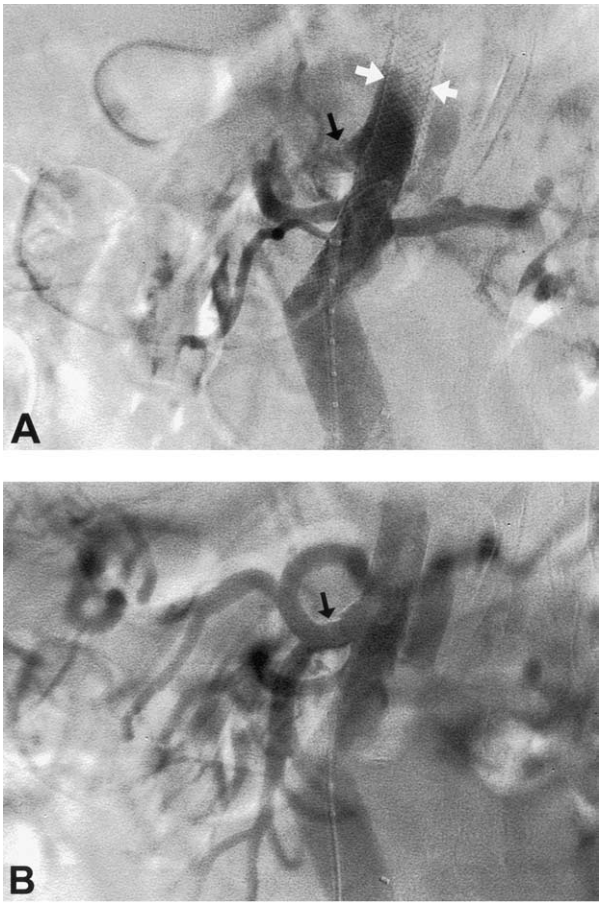

Fig 3. A, Aortogram demonstrates aortic dissection with a stent in the true lumen *(white arrows)* and poor filling of the celiacomensenteric trunk *(black arrow)* from the false lumen. **B,** After endovascular fenestration of the septum between the true and false lumens, improved flow through the celiacomensenteric trunk is evident *(black arrow)*.

(n = 3), evaluation of renal vessels for donor nephrectomy (n = 2), evaluation for correctable causes of hypertension in a patient with a known abdominal aortic aneurysm (n = 1), and lower extremity rest pain (n = 1). The fourteenth CMT was found intraoperatively at pancreaticoduodenectomy. None of these patients had intrinsic aneurysmal or occlusive disease involving the CMT, although 1 patient had an embolus from an atrial source lodged in the CMT that required systemic anticoagulation, and another patient had a thoracic aortic dissection that extended into the CMT without any compromise of the arterial lumen.

DISCUSSION

Splanchnic arteries develop during the fourth week of gestation, with the formation of the paired vitelline or ventral segmental arteries originating from the 2 dorsal aortae.[4] The dorsal aortae and ventral segmental roots then fuse, resulting in the midline vessels. The foregut and midgut derive their blood supply from the 10th through 13th ventral segmental arteries. A longitudinal arterial anastomosis that runs parallel to the aorta connects these roots during this period of fetal development (Fig 4, A). The 11th and 12th ventral segmental arteries along with the longitudinal anastomosis eventually regress, leaving the 10th segmental artery to become the celiac artery and the 13th segmental artery to become the superior mesenteric artery (Fig 4, B). Variations in celiac and mesenteric anatomy are thought to occur as a result of alterations in the regression of the 10th through 12th ventral segmental roots with persistence of the longitudinal anastomotic artery. This leaves the 13th ventral segmental root as the common origin for both the celiac and superior mesenteric arteries (Fig 4, C). The proximal course of the CMT was horizontal in this series of patients, perhaps reflecting a more caudal origin of the vessel than usually observed with the celiac artery, which in nearly 20% of the population has an acute aortic origin associated with its passage beneath the media arcuate ligament. It is interesting that CMT entrapment at the aortic hiatus by this ligamentous tissue has not been reported.

Splanchnic artery aneurysms have been reported in approximately 0.2% of the population, with most located in the hepatic and splenic arteries. Six case reports of aneurysmal dilation of a CMT have been described in the literature; all were successfully treated with operative intervention, including the first reported case, which was treated at our institution.[6-11] Persistent embryonic arteries may be more prone to congenital defects in their arterial elastic and smooth muscle layers. This may predispose those vessels to aneurysm formation. This is well recognized in the case of persistent sciatic artery, which often becomes aneurysmal. In this regard the CMT, an embryologic error, may be at increased risk for aneurysm formation.

An occluded CMT has not been previously reported. Occlusive disease of a CMT would logically produce symptoms of acute or chronic mesenteric ischemia, as was evident in the present patient series. The redundancy between the celiac and superior mesenteric arterial circulation is nonexistent in the case of a CMT, and a proximal stenosis affecting this vessel may have serious ischemic consequences to the intestines. It is in this setting that endovascular interventions in the CMT present considerable therapeutic risks.

Aortic dissections involving a CMT may reflect an underlying congenital defect of the arterial wall. The CMT was an unlikely entry point in aortic dissections in this series. It is reasonable to speculate that a CMT may be a marker for increased risk for aortic dissection, as is a bicuspid aortic valve.

An understanding of the clinical relevance of CMT lesions is likely to remain anecdotal, given the infrequency of this anomaly. Nevertheless, it appears that most incidentally diagnosed CMTs without intrinsic disease will remain asymptomatic. Although many CMTs are first incidentally recognized during studies of unrelated symptoms, some may have clinically relevant aneurysm or occlusive disease that warrants operative correction. In these selected pa-

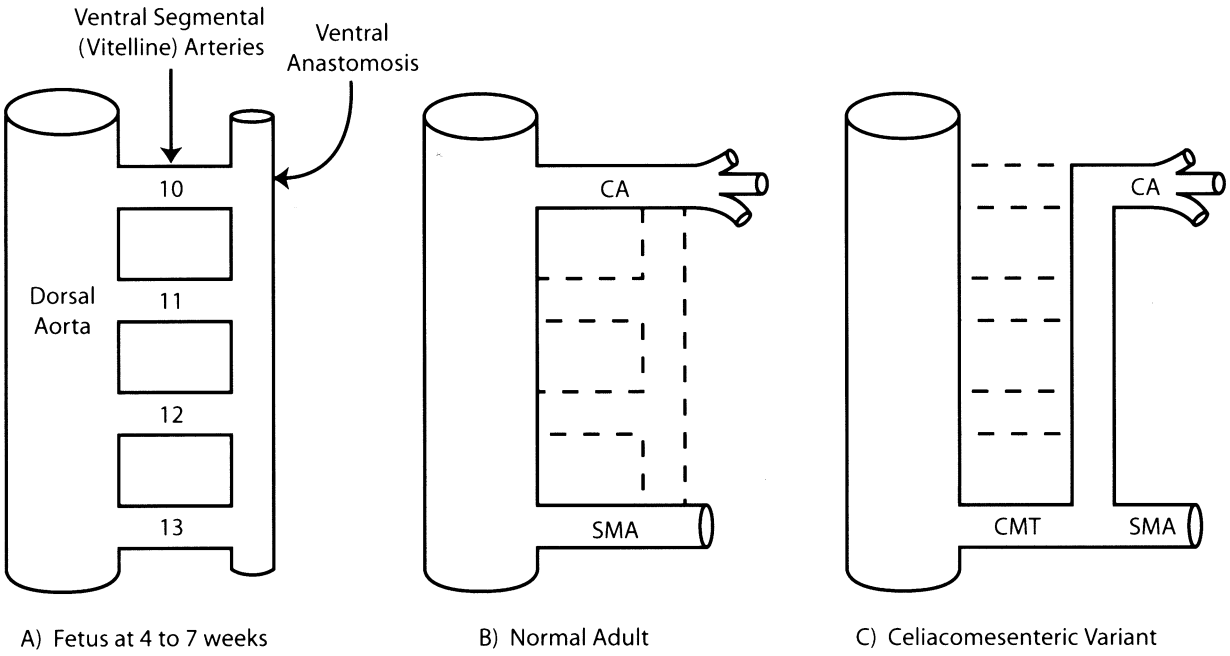

Fig 4. A, Illustration of dorsal aorta, ventral segmental roots 10 through 13, and longitudinal anastomosis at 4 to 7 weeks of fetal development. **B,** Normal development in adult splanchnic vessels after regression of roots 11 and 12, and the longitudinal anastomosis; the celiac artery *(CA)* and superior mesenteric artery *(SMA)* are derived from roots 10 and 13, respectively. **C,** Celiacomesenteric variant with development of a celiacomensenteric trunk *(CMT)* follows regression of roots 10, 11, and 12, with persistence of the longitudinal anastomosis. *CA,* Celiac artery; *SMA,* superior mesenteric artery.

tients, carefully planned and executed operative intervention seems appropriate.

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